Amendments to the Specification:

Please amend the paragraph from page 57, line 17 through page 58, page 59, lines 1-2 as follows:

In this study, we further characterize the biological significance of VCPs ability to bind heparin. Using flow cytometry, the amount of specific antibody binding to human endothelial cells – in the presence and absence of VCP – was measured. It was found that VCP was able to inhibit antibody binding to major histocompatibility complex class I molecules on [[hu]] human endothelial cells. This suggests that VCP can interfere with molecular interactions with infected cells and could prevent antibody-dependent cell-mediated cytotoxicity as well as other cytotoxic cell interactions with target cells. The ability of VCP to bind heparin-like molecules suggests that it plays many roles and therefore may have a variety of applications. It is for these reasons that we have been interested in obtaining a better understanding of the molecular basis for the VCP-heparin interaction. Through examination of several recombinant VCP (rVCP) fragments, it has now been determined that the percentage of positively charged amino acids, overall charge, and the number of putative heparin binding sites are all important factors governing the heparin binding ability of VCP.

Flow microfluorimetric analysis. Human umbilical cord vascular endothelial cells (HUVECs) were obtained from the American Type Culture Collection (Manassas, VA) at passage thirteen. Monolayer cultures were maintained using F12K Ham's media supplemented with 10% fetal bovine serum (FBS), (Sigma), 30 μg/ml endothelial cell growth supplement (Sigma), and 100 μg/ml heparin (Sigma), at 37°C in humidified air containing 5% CO_2. Cells were cultured to approximately 80% confluency in 75 cm^2 vented flasks (Flacon, Lincoln Park, NJ) coated with 1.5% gelatin (Sigma) in phosphate buffer saline. Cells were trypsinized (0.25% trypsin, 1 mM EDTA, Sigma), 4 X 10^5 cells/ml were placed in a 6-well flat-bottom culture plates (3.5 cm diameter, Falcon, Lincoln Park, NJ), coated with 1.5 % gelatin, and incubated for 24 h in F12K media without growth factor (Lian et al., 1996. *J. Immunol.* 157:864-873). For analysis of antibody interaction with cell surface class I HLA-ABC molecules, triplicate wells (2 ml each) were trypsinized and washed with FTA hemagglutination buffer (Becton Dickinson) and stained for 30 min on ice with 0.25 μg of phycoerythrin-conjugated mouse antihuman HLA-ABC monoclonal antibody (Caltag, Burlingame, CA), or a mouse IgG2a mAb (an isotype-match

negative control) in the presence or absence of 2 or 5 μg of VCP. After incubation, cells were washed three times in FTA buffer, and then fixed in Hank's balanced salt solution (HBSS) containing 2% paraformaldehyde. Before staining, cell cultures were assessed for viability by trypan blue dye exclusion and, in all cases, the cells were found to be >95% viable. The percentage of positively stained cells were determined using a flow cytometer (Becton Dickinson FACScan, San Jose, CA) equipped with a single 15-mW argon laser tuned to 488 nm. Forward and 90° angle light scatter and integrated log phycoerythrin and FITC fluorescence signals were collected and analysed. Variability between duplicate samples was less than 10%. To compensate for any background fluorescence, the control threshold was set less than 1% binding of control mAbs. Data were acquired from analysis of >3000 events. A single homogenous cell population was indicated as detected by forward and 90° light scatter.